```
 NEWS 25   APR 30  CHEMCATS enhanced with 1.2 million new records
 NEWS 26   APR 30  CA/CAplus enhanced with 1870-1889 U.S. patent
records
 NEWS 27   APR 30  INPADOC replaced by INPADOCDB on STN
 NEWS 28   MAY 01  New CAS web site launched
 NEWS 29   MAY 08  CA/CAplus Indian patent publication number format
defined
 NEWS 30   MAY 14  RDISCLOSURE on STN Easy enhanced with new search
and display
                   fields
 NEWS 31   MAY 21  BIOSIS reloaded and enhanced with archival data
 NEWS 32   MAY 21  TOXCENTER enhanced with BIOSIS reload
 NEWS 33   MAY 21  CA/CAplus enhanced with additional kind codes for
German
                   patents
 NEWS 34   MAY 22  CA/CAplus enhanced with IPC reclassification in
Japanese
                   patents

 NEWS EXPRESS   NOVEMBER 10 CURRENT WINDOWS VERSION IS V8.01c, CURRENT
                MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
                AND CURRENT DISCOVER FILE IS DATED 25 SEPTEMBER 2006.

 NEWS HOURS     STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN     Welcome Banner and News Items
 NEWS IPC8      For general information regarding STN implementation
of IPC 8

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * *
* *

FILE 'HOME' ENTERED AT 16:24:14 ON 11 JUN 2007

=> File Medline EMBASE Biosis Caplus
COST IN U.S. DOLLARS                                 SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'MEDLINE' ENTERED AT 16:24:58 ON 11 JUN 2007

FILE 'EMBASE' ENTERED AT 16:24:58 ON 11 JUN 2007
Copyright (c) 2007 Elsevier B.V. All rights reserved.
```

```
FILE 'BIOSIS' ENTERED AT 16:24:58 ON 11 JUN 2007
Copyright (c) 2007 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 16:24:58 ON 11 JUN 2007
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2007 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (casein kinase) (w) (I or 1)
L1         2877 (CASEIN KINASE) (W) (I OR 1)

=> s (inhibitor or inhibitors)
L2      2755119 (INHIBITOR OR INHIBITORS)

=> s L1 (4A) l2
L3          136 L1 (4A) L2

=> s l2 (6A) cell or cellular
L4      1591079 L2 (6A) CELL OR CELLULAR

=> s l3 and l4
L5           17 L3 AND L4

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):15
DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L5
L6            10 DUPLICATE REMOVE L5 (7 DUPLICATES REMOVED)

=> d l6 1-10 bib ab

L6   ANSWER 1 OF 10  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2006:13464  CAPLUS
DN   144:101073
TI   therapeutic uses of kinase inhibitors, and compositions thereof
IN   Caligiuri, Maureen G.; Kley, Nikolai A.; Murthi, Krishna K.
PA   GPC Biotech, Inc., USA
SO   PCT Int. Appl., 201 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.
DATE
     ---------------     ----   --------    --------------------
--------
PI   WO 2006002119        A2    20060105    WO 2005-US21843
20050617
```

WO 2006002119 A3 20070222
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ,
LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA,
NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK,
SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU,
ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF,
CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM,
KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG,
KZ, MD, RU, TJ; TM
EP 1763345 A2 20070321 EP 2005-762859 20050617
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA,
HR, LV, MK, YU
PRAI US 2004-580868P P 20040618
WO 2005-US21843 W 20050617
OS MARPAT 144:101073
AB The invention pertains to inhibitors of various kinases (e.g. S/T kinases,
Tyr kinases, etc.), which inhibitors are previously known as cyclin
dependent kinase inhibitors (CDKs). The inhibitors of the invention are
capable of inhibiting various wild-type and mutant form kinases, including
drug-resistant forms of mutant kinases. Thus, the kinase inhibitors are
useful in treating a wide range of diseases/conditions associated with
abnormal functions/excessive activities of the target kinases, including
mutant kinases. The invention further provides methods for treating
cancers, tumors and patients which are resistant or refractory to other

therapeutic agents. Pharmaceutical compns. and packaged pharmaceuticals
with instructions of these inhibitors, and methods of using these inhibitors are also provided.

L6 ANSWER 2 OF 10 MEDLINE on STN DUPLICATE 1
AN 2006124588 MEDLINE
DN PubMed ID: 16247451
TI RNAi-based screening of the human kinome identifies Akt-cooperating
kinases: a new approach to designing efficacious multitargeted kinase
inhibitors.
AU Morgan-Lappe S; Woods K W; Li Q; Anderson M G; Schurdak M E; Luo Y;
Giranda V L; Fesik S W; Leverson J D
CS Abbott Laboratories, Cancer Research, Abbott Park, IL 60064, USA.
SO Oncogene, (2006 Mar 2) Vol. 25, No. 9, pp. 1340-8.
Journal code: 8711562. ISSN: 0950-9232.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200604
ED Entered STN: 3 Mar 2006
Last Updated on STN: 19 Apr 2006
Entered Medline: 18 Apr 2006
AB Tumors comprise genetically heterogeneous cell populations, whose growth
and survival depend on multiple signaling pathways. This has spurred the
development of multitargeted therapies, including small molecules that can
inhibit multiple kinases. A major challenge in designing such molecules
is to determine which kinases to inhibit in each cancer to maximize
efficacy and therapeutic index. We describe an approach to this problem
implementing RNA interference technology. In order to identify
Akt-cooperating kinases, we screened a library of kinase-directed small
interfering RNAs (siRNAs) for enhanced cancer cell killing in
the presence of Akt inhibitor A-443654. siRNAs targeting
casein kinase I gamma 3 (CSNK1G3) or the
inositol polyphosphate multikinase (IPMK) significantly enhanced
A-443654-mediated cell killing, and caused decreases in Akt Ser-473 and
ribosomal protein S6 phosphorylation. Small molecules targeting CSNK1G3

and/or IPMK in addition to Akt may thus exhibit increased efficacy and
have the potential for improved therapeutic index.

L6 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:451232 CAPLUS
DN 143:19954
TI Methods for inhibiting cell growth
IN Zhao, Yi; Chandraratna, Roshantha A.
PA Allergan, Inc., USA
SO PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005046726	A2	20050526	WO 2004-US37881	20041112
WO 2005046726	A3	20051208		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2003-519528P P 20031112
US 2004-564807P P 20040422

AB Cell growth is inhibited and/or cell death is induced in a cell by
administering an RXR (retinoid X receptor) agonist and an inhibitor of casein kinase 1
α. A cell or a tissue can be screened for enhanced susceptibility

to cell death or interference with cell growth. Conditions characterized
by uncontrolled cell growth or proliferation, such as a cancer, can be
treated with inhibitors of casein kinase
1 α.

L6 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:521462 CAPLUS
DN 137:88442
TI Incensole and furanogermacrens and compounds in treatment for inhibiting
neoplastic lesions and microorganisms
IN Shanahan-Pendergast, Elisabeth
PA Ire.
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002053138	A2	20020711	WO 2002-IE1	20020102
WO 2002053138	A3	20020919		
W: AE, AG, AT, AU, BB, BG, CA, CH, CN, CO, CU, CZ, LU, LV, MA, MD, UA, UG, US, VN, YU, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, AT, BE, CH, CY, DE, ES, FI, ML, MR, NE, SN, TD, TG				
AU 2002219472	A1	20020716	AU 2002-219472	20020102
EP 1351678	A2	20031015	EP 2002-727007	20020102
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2004092583	A1	20040513	US 2004-250535	20040102
PRAI IE 2001-2	A	20010102		
WO 2002-IE1	W	20020102		

OS MARPAT 137:88442
AB The invention discloses the use of incensole and/or furanogermacrens,
derivs. metabolites and precursors thereof in the treatment of neoplasia,
particularly resistant neoplasia and immunodysregulatory disorders. These

compds. can be administered alone or in combination with conventional chemotherapeutic, antiviral, antiparasite agents, radiation and/or surgery. Incensole and furanogermacren and their mixture showed antitumor activity against various human carcinomas and melanomas and antimicrobial activity against Staphylococcus aureus and Enterococcus faecalis.

L6 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:432817 CAPLUS
DN 135:41041
TI Use of hymenialdisine or a derivative thereof as an inhibitor of cyclin-dependent kinases, GSK-3β and casein kinase 1, and therapeutic use
IN Meijer, Laurent
PA Centre National de la Recherche Scientifique (CNRS), Fr.
SO Eur. Pat. Appl., 38 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1106180	A1	20010613	EP 1999-403077	19991208
EP 1106180	B1	20031112		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
AT 253918	T	20031115	AT 1999-403077	19991208
ES 2213996	T3	20040901	ES 1999-403077	19991208
CA 2384982	A1	20010614	CA 2000-2384982	20001207
WO 2001041768	A2	20010614	WO 2000-EP12791	20001207
WO 2001041768	A3	20020510		
WO 2001041768	A9	20020912		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,				

SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
EP 1235578 A2 20020904 EP 2000-987404 20001207
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2004500356 T 20040108 JP 2001-543113 20001207
US 2003105075 A1 20030605 US 2002-149115 20021004
US 7098204 B2 20060829
PRAI EP 1999-403077 A 19991208
WO 2000-EP12791 W 20001207
AB The title compds. are I (R1, R2 = H, Br), or a pharmaceutically acceptable
salt thereof, are used for the manufacture of a medicament for inhibiting
cyclin-dependent kinases, GSK-3β, and casein kinase 1. The compds.
may be used for preventing and treating neurodegenerative disorders (e.g.
Alzheimer's disease), diabetes, inflammatory pathologies, and cancers.
RE.CNT 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:856756 CAPLUS
DN 134:129061
TI IC261, a specific inhibitor of the protein kinases casein kinase 1-delta and -epsilon, triggers
the mitotic checkpoint and induces p53-dependent postmitotic effects
AU Behrend, L.; Milne, D. M.; Stoter, M.; Deppert, W.; Campbell, L. E.; Meek,
D. W.; Knippschild, U.
CS Heinrich-Pette-Institut fur Experimentelle Virologie und Immunologie,
Hamburg, D-20251, Germany
SO Oncogene (2000), 19(47), 5303-5313
CODEN: ONCNES; ISSN: 0950-9232
PB Nature Publishing Group
DT Journal
LA English

AB The p53-targeted kinases casein kinase 1δ (CK1δ) and casein kinase 1ε (CK1ε) have been proposed to be involved in regulating DNA repair and chromosomal segregation. Recently, we showed that CK1δ localizes to the spindle apparatus and the centrosomes in cells with mitotic failure caused by DNA-damage prior to mitotic entry. We provide here evidence that 3-(2,4,6-trimethoxyphenyl)methylidenyl-indolin-2-one (IC261), a novel inhibitor of CK1δ and CK1ε, triggers the mitotic checkpoint control. At low micromolar concns. IC261 inhibits cytokinesis causing a transient mitotic arrest. Cells containing active p53 arrest in the postmitotic G1 phase by blockage of entry into the S phase. Cells with non-functional p53 undergo postmitotic replication developing an 8N DNA content. The increase of DNA content is accompanied by a high amount of micronucleated and apoptotic cells. Immunfluorescence images show that at low concns. IC261 leads to centrosome amplification causing multipolar mitosis. Our data are consistent with a role for CK1δ and CK1ε isoforms in regulating key aspects of cell division, possibly through the regulation of centrosome or spindle function during mitosis.

RE.CNT 67 THERE ARE 67 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 10 MEDLINE on STN DUPLICATE 2
AN 1998366074 MEDLINE
DN PubMed ID: 9700717
TI H-7-induced apoptosis in the cells of a Drosophila neuronal cell line through affecting unidentified H-7-sensitive substance(s).
AU Nagano M; Suzuki H; Ui-Tei K; Sato S; Miyake T; Miyata Y
CS Department of Pharmacology, Nippon Medical School, Tokyo, Japan.
SO Neuroscience research, (1998 Jun) Vol. 31, No. 2, pp. 113-21.
Journal code: 8500749. ISSN: 0168-0102.
CY Ireland
DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199811

ED Entered STN: 6 Jan 1999
Last Updated on STN: 6 Jan 1999
Entered Medline: 20 Nov 1998
AB The present study was undertaken to reveal underlying mechanisms of
apoptosis in neurons using clonal neuronal cells, ML-DmBG2-c2, derived
from Drosophila larval central nervous system
1-(5-Isoquinolinesulfonyl)-2-
methylpiperazine (H-7), a protein kinase inhibitor, induced
cell death with typical features of apoptosis such as
internucleosomal DNA fragmentation, nuclear condensation and apoptotic
bodies in the cells. Though H-7 is known to inhibit cAMP-dependent
protein kinase (PKA), protein kinase C (PKC), cGMP-dependent protein
kinase (PKG), myosin light chain kinase (MLCK), and casein
kinase I (CKI), specific inhibitors for these
kinases such as H-89, calphostin C, ML-9, or CKI-7 did not induce
apoptosis in the cells. Other kinases such as tyrosine kinase.
PI3-kinase and Ca2+/CaM kinase II so far examined in the present study
were interpreted not to be involved in the apoptotic cascade. Therefore,
it is concluded that an H-7-sensitive substance(s) other than these
kinases is responsible for the apoptosis in the neuronal cells. Caspase
inhibitors prevented apoptosis in the cells treated with H-7. These
results suggest that caspase(s) is involved downstream of the
H-7-sensitive point in the cascade of the apoptosis.

L6 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:301667 CAPLUS
DN 122:127030
TI Development of inhibitors of protein kinases CKI and CKII and some related
aspects, including donor and acceptor specificities and viral protein
kinases
AU Shugar, David
CS Inst. Biochem. Biophysics, Polish Academy Sciences, Warszawa, 02-106, Pol.
SO Cellular & Molecular Biology Research (1994), 40(5/6), 411-19
CODEN: CMBREW; ISSN: 0968-8773
PB Elsevier
DT Journal; General Review
LA English
AB A review with .apprx.45 refs. A brief overview is presented of progress

in the development of specific inhibitors of protein kinases CKI and CKII.
Two promising classes of inhibitors, which have the ability to traverse cell membranes, are now known. One of these is based on halogenated benzimidazoles and 2-aza-benzimidazoles (benzotriazoles)
and some of their nucleosides. The second embraces modified isoquinoline
sulfonamides, several of which are known as inhibitors of other protein
kinases. Both classes include analogs that permit discrimination between
CKI and CKII. Ongoing research with halogenated benzotriazoles leads to
inhibitors with Ki values below 1 μM. Also considered are nucleoside
triphosphate analog inhibitors and their potential properties as donors,
with illustrative examples from the field of nucleoside kinases, including
the apparent existence of a dual-specific viral protein/nucleoside kinase.
The role of cellular CKII and viral-encoded CKII-like activities in viral replication underlines the potential of CKII inhibitors as
antiviral agents, exemplified by the case of vesicular stomatitis virus.

L6 ANSWER 9 OF 10 MEDLINE on STN DUPLICATE 3
AN 91120135 MEDLINE
DN PubMed ID: 2278876
TI A protein complex expressed during terminal differentiation of monomyelocytic cells is an inhibitor of cell growth.
AU Murao S; Collart F; Huberman E
CS Biological and Medical Research Division, Argonne National Laboratory,
Illinois 60439.
SO Cell growth & differentiation : the molecular biology journal of the
American Association for Cancer Research, (1990 Oct) Vol. 1, No. 10, pp.
447-54.
Journal code: 9100024. ISSN: 1044-9523.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
LA English
FS Priority Journals
EM 199103
ED Entered STN: 5 Apr 1991
Last Updated on STN: 3 Feb 1997

Entered Medline: 12 Mar 1991
AB A protein complex (PC) composed of the MRP8 and MRP14 proteins has previously been shown to be a specific inhibitor of casein kinase I and II. This PC is expressed during the late stages of terminal differentiation induced in human promyelocytic HL-60 leukemia cells by 1 alpha,25-dihydroxyvitamin D3 and in human monocytic THP-1 leukemia cells by phorbol 12-myristate 13-acetate. This expression is associated with terminal cell differentiation because incubation of HL-60 cells with an agent or condition that causes suppression of growth but not induction of differentiation does not result in expression of the PC. At concentrations of 5-15 nM, the purified PC inhibited the growth of HL-60 cells and THP-1 cells, as well as other cell types belonging to different cell lineages. This growth inhibition was preceded by a reduction in [32P]phosphate incorporation and, at the higher PC concentrations, was associated with a reduction in [3H]thymidine, [3H]uridine, and [32S]methionine incorporation. The specific expression pattern and growth-inhibitory character of the PC suggests that the complex may have a role in suppressing cell growth during monomyelocytic terminal differentiation induced by specific chemical stimuli and during physiological and pathological events associated with monomyelocytic cell functions.

L6 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1990:607124 CAPLUS
DN 113:207124
TI Casein kinase 2: an 'eminence grise' in cellular regulation?
AU Pinna, Lorenzo A.
CS Dip. Chim. Biol., Univ. Padova, Padua, 35121, Italy
SO Biochimica et Biophysica Acta, Molecular Cell Research (1990), 1054(3), 267-84
CODEN: BBAMCO; ISSN: 0167-4889
DT Journal; General Review
LA English
AB A review, with 176 refs., on casein kinase 2 (CK2) with emphasis on the features of CK2, subunit composition, structure of the α- and β-subunits, regulation of CK2, biol. functions, phosphorylatable substrates, substrate and inhibitor specificity, and comparison

to casein kinase 1.